What is claimed is:

1. A method for reducing levels of Aβ peptide in a mammal, comprising administering a therapeutically effective amount of a soluble Nogo receptor polypeptide.

2. The method of claim 1, wherein the levels of Aβ peptide are elevated in association with a disease, disorder or condition.

3. The method of claim 2, wherein said disease, disorder or condition is Alzheimer's disease.

4. The method of claim 1, wherein the soluble Nogo receptor polypeptide is administered by bolus injection or chronic infusion.

5. The method of claim 4, wherein the soluble Nogo receptor polypeptide is administered intravenously.

6. The method of claim 4, wherein the soluble Nogo receptor polypeptide is administered directly into the central nervous system.

7. The method of claim 6, wherein the soluble Nogo receptor polypeptide is administered directly into a lateral ventricle.

8. The method of any one of claims 1-3, wherein the soluble Nogo receptor polypeptide is a soluble form of a mammalian NgR1.

9. The method of claim 8, wherein the soluble form of a mammalian NgR1: (a) comprises amino acids 26 to 310 of human NgR1 (SEQ ID NO: 3) with up to ten conservative amino acid substitutions; and (b) lacks (i) a functional transmembrane domain, and (ii) a functional signal peptide.

10. The method of claim 8, wherein the soluble form of a mammalian NgR1: (a) comprises amino acids 26 to 344 of human NgR1 (SEQ ID NO: 4) with up to ten

conservative amino acid substitutions; and (b) lacks (i) a functional transmembrane domain, and (ii) a functional signal peptide.

11. The method of claim 8, wherein the soluble form of a mammalian NgR1: (a) comprises amino acids 27 to 310 of rat NgR1 (SEQ ID NO: 5) with up to ten conservative amino acid substitutions; and (b) lacks (i) a functional transmembrane domain, and (ii) a functional signal peptide.

12. The method of claim 8, wherein the soluble form of a mammalian NgR1: (a) comprises amino acids 27 to 344 of rat NgR1 (SEQ ID NO: 6) with up to ten conservative amino acid substitutions; and (b) lacks (i) a functional transmembrane domain, and (ii) a functional signal peptide.

13. The method of claim 8, wherein the soluble form of a mammalian NgR1 further comprises a fusion moiety.

14. The method of claim 13, wherein the fusion moiety is an immunoglobulin moiety.

15. The method of claim 14, wherein the immunoglobulin moiety is an Fc moiety.

16. The method of any one of claims 1-3, wherein the therapeutically effective amount is from 0.001 mg/kg to 10 mg/kg.

17. The method of claim 16, wherein the therapeutically effective amount is from 0.01 mg/kg to 1.0 mg/kg.

18. The method of claim 17, wherein the therapeutically effective amount is from 0.05 mg/kg to 0.5 mg/kg.

19. A method of preventing or treating a disease, disorder or condition associated with plaques of Aβ peptide in a mammal, comprising administering a therapeutically effective amount of an NgR1 antagonist.

20. The method of claim 19, wherein said plaques are in the central nervous system.

21. The method of claim 20, wherein said disease, disorder or condition is Alzheimer's Disease.

22. The method of any one of claims 19-21, wherein the NgR1 antagonist is administered directly into the central nervous system.

23. The method of claim 22, wherein the NgR1 antagonist is administered directly into the a lateral ventricle.

24. The method of claim 22, wherein the NgR1 antagonist is administered by bolus injection or chronic infusion.

25. The method of any one of claims 19-21, wherein the NgR1 antagonist comprises a soluble form of a mammalian NgR1.

26. The method of claim 25, wherein the soluble form of a mammalian NgR1: (a) comprises amino acids 26 to 310 of human NgR1 (SEQ ID NO: 3) with up to ten conservative amino acid substitutions; and (b) lacks (i) a functional transmembrane domain, and (ii) a functional signal peptide.

27. The method of claim 25, wherein the soluble form of a mammalian NgR1: (a) comprises amino acids 26 to 344 of human NgR1 (SEQ ID NO: 4) with up to ten conservative amino acid substitutions; and (b) lacks (i) a functional transmembrane domain, and (ii) a functional signal peptide.

28. The method of claim 25, wherein the soluble form of a mammalian NgR1: (a) comprises amino acids 27 to 310 of rat NgR1 (SEQ ID NO: 5) with up to ten conservative amino acid substitutions; and (b) lacks (i) a functional transmembrane domain, and (ii) a functional signal peptide.

29. The method of claim 25, wherein the soluble form of a mammalian NgR1: (a) comprises amino acids 27 to 344 of rat NgR1 (SEQ ID NO: 6) with up to ten conservative amino acid substitutions; and (b) lacks (i) a functional transmembrane domain, and (ii) a functional signal peptide.

30. The method of claim 25, wherein the soluble form of a mammalian NgR1 further comprises a fusion moiety.

31. The method of claim 30, wherein the fusion moiety is an immunoglobulin moiety.

32. The method of claim 31, wherein the immunoglobulin moiety is an Fc moiety.

33. The method of any one of claims 19-21, wherein the NgR1 antagonist comprises an antibody or antigen-binding fragment thereof that binds to a mammalian NgR1.

34. The method of claim 33, wherein the antibody is selected from the group consisting of a polyclonal antibody, a monoclonal antibody, a Fab fragment, a Fab' fragment, a $F(ab')_2$ fragment, an Fv fragment, an Fd fragment, a diabody, and a single-chain antibody.

35. The method of claim 33, wherein the antibody or antigen-binding fragment thereof binds to an polypeptide bound by a monoclonal antibody produced by a hybridoma selected from the group consisting of: HB 7E11 (ATCC® accession No. PTA-4587), HB 1H2 (ATCC® accession No. PTA-4584), HB 3G5 (ATCC® accession No. PTA-4586), HB 5B10 (ATCC® accession No. PTA-4588) and HB 2F7 (ATCC® accession No. PTA-4585).

36. The method of claim 35, wherein said monoclonal antibody is produced by the HB 7E11 hybridoma.

37. The method of claim 36, wherein the polypeptide comprises an amino acid sequence selected from the group consisting of: AAAFGLTLLEQLDLSDNAQLR (SEQ

ID NO: 7); LDLSDNAQLR (SEQ ID NO: 8); LDLSDDAELR (SEQ ID NO: 9); LDLASDNAQLR (SEQ ID NO: 10); LDLASDDAELR (SEQ ID NO: 11); LDALSDNAQLR (SEQ ID NO: 12); LDALSDDAELR (SEQ ID NO: 13); LDLSSDNAQLR (SEQ ID NO: 14); LDLSSDEAELR (SEQ ID NO: 15); DNAQLRVVDPTT (SEQ ID NO: 16); DNAQLR (SEQ ID NO: 17); ADLSDNAQLRVVDPTT (SEQ ID NO: 18); LALSDNAQLRVVDPTT (SEQ ID NO: 19); LDLSDNAALRVVDPTT (SEQ ID NO: 20); LDLSDNAQLHVVDPTT (SEQ ID NO: 21); and LDLSDNAQLAVVDPTT (SEQ ID NO: 22).

38. The method of claim 36, wherein the polypeptide consists of an amino acid sequence selected from the group consisting of: AAAFGLTLLEQLDLSDNAQLR (SEQ ID NO: 7); LDLSDNAQLR (SEQ ID NO: 8); LDLSDDAELR (SEQ ID NO: 9); LDLASDNAQLR (SEQ ID NO: 10); LDLASDDAELR (SEQ ID NO: 11); LDALSDNAQLR (SEQ ID NO: 12); LDALSDDAELR (SEQ ID NO: 13); LDLSSDNAQLR (SEQ ID NO: 14); LDLSSDEAELR (SEQ ID NO: 15); DNAQLRVVDPTT (SEQ ID NO: 16); DNAQLR (SEQ ID NO: 17); ADLSDNAQLRVVDPTT (SEQ ID NO: 18); LALSDNAQLRVVDPTT (SEQ ID NO: 19); LDLSDNAALRVVDPTT (SEQ ID NO: 20); LDLSDNAQLHVVDPTT (SEQ ID NO: 21); and LDLSDNAQLAVVDPTT (SEQ ID NO: 22).

39. The method of any one of claims 19-21, wherein the therapeutically effective amount is from 0.001 mg/kg to 10 mg/kg.

40. The method of claim 39, wherein the therapeutically effective amount is from 0.01 mg/kg to 1.0 mg/kg.

41. The method of claim 40, wherein the therapeutically effective amount is from 0.05 mg/kg to 0.5 mg/kg.